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Protective Factors in the Association Between Child Sexual Abuse and Telomere Length in  
Adults

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science  
at Virginia Commonwealth University.

by

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## Abstract

PROTECTIVE FACTORS IN THE ASSOCIATION BETWEEN CHILD SEXUAL ABUSE  
AND TELOMERE LENGTH IN ADULTS

By David W. Sosnowski, B.S.

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science  
at Virginia Commonwealth University

Virginia Commonwealth University, 2017

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The purpose of the present study was to examine if childhood sexual abuse (CSA) was associated with decreases in mean telomere length (TL), and if social support and/or optimism moderated this association. The study included 99 Caucasian female monozygotic twins, ranging in age from 19-48 ( $M_{\text{age}} = 30.5$ ,  $SD = 7.8$ ) at Time 1. Linear mixed effects models were employed to test study hypotheses. Analyses with all participants did not detect an effect of CSA exposure or severity on mean TL, nor were there effects with optimism. However, in analyses that only included women exposed to abuse, increases in social support were associated with increases in mean TL. Further, for women who experienced non-genital abuse, social support was positively associated with mean TL. Findings from the current study clarify the role of CSA in telomere attrition, and factors that may protect against the negative biological effects of CSA.

## Protective Factors in the Association Between Child Sexual Abuse and Telomere Length in Adults

Child sexual abuse (CSA) continues to be a pervasive issue with both short- and long-term physical and psychological consequences for its victims (Browne & Finkelhor, 1986; Irish, Kobayashi, Douglas, & Delahanty, 2009; Polusny & Follette, 1995; Wegman & Stetler, 2009). According to Butchart and colleagues (2006), sexual abuse is defined as “the involvement of a child in sexual activity that he or she does not fully comprehend, is unable to give informed consent to, or for which the child is not developmentally prepared, or else that violates the laws or social taboos of society” (p. 10). According to a recent report by the World Health Organization (WHO), 1 in 5 women and 1 in 13 men worldwide report having been sexually abused as children (WHO, 2016). In the United States, estimates reveal that 27% of females report experiencing sexual abuse by age 17, while just over 5% of males report experiencing sexual abuse by this age (Finkelhor, Shattuck, Turner, & Hamby, 2014). Although the psychological consequences of CSA have been well documented (for a list of reviews, see Maniglio, 2009), the physical health consequences of CSA – and specifically the mechanisms linking CSA to physical health – have received less attention (Wegman & Stetler, 2009).

Several theories explaining how ACEs – particularly CSA – affect development and long-term physical health have been proposed. For example, Masten and Cicchetti (2010) posit that ACEs set in motion a variety of interrelated events over time (i.e., a developmental cascade) that ultimately leads to poor mental and/or physical health outcomes later in life. Similarly, Miller and colleagues (2011) hypothesize that ACEs lead to various biological and epigenetic alterations that become programmed into an individual and contribute to the development of chronic diseases of aging (e.g., cardiovascular disease). Lastly, various neurobiological theories

have been proposed, suggesting that the severe and chronic stress associated with CSA causes a prolonged increase in the body's stress response, which over time can lead to maladaptation of the HPA axis and other physiological processes that contribute to physical health problems (cf, Hulme, 2004; Masten & Cicchetti, 2010; Miller, Chen, & Parker, 2011). Although each of these theories explains how CSA can affect development and impair physical health in adulthood, the mechanisms that ultimately contribute to poor health in adulthood are still under debate. Recent theoretical and empirical advances, however, have suggested that alterations in biological functioning may play a pivotal role in long-term physical health outcomes for individuals exposed to early life adversity (e.g., Miller et al., 2009; Miller et al., 2011).

### **Biological Programming and Telomeres**

Understanding how ACEs influence biological functioning is necessary because it offers one explanation for how certain social experiences can “get under the skin” and become programmed into an individual, with effects lasting well after the event has occurred. The concept of biological programming was introduced with Barker's (1990) fetal programming hypothesis. This hypothesis postulates that maternal stress during pregnancy increases cortisol concentrations in the fetus, which can impact physiological functioning of the child in utero and have effects on postnatal outcomes such as low birth weight (Graignic-Philippe, Dayan, Chokron, Jacquet, & Tordjman, 2014), high stress reactivity during infancy (Oberlander et al., 2008), and emotional and behavioral problems throughout childhood (Talge, Neal, & Glover, 2007). Similarly, it is possible that adversity experienced in the postnatal environment can become programmed into an individual and impact biological processes that contribute to adult diseases (e.g., Felitti et al., 1998; Miller et al., 2011).

Telomere attrition is one biological process that recently has received much attention in the literature because it has been linked to various types of psychosocial stressors and diseases later in life (Oliveira et al., 2016; Rode et al., 2015). Telomeres are protein-bound DNA structures (in humans, TTAGGG) located at the end of chromosomes (Blackburn, 2005; see Figure 1). Their primary functions are to regulate cellular replication and prevent the loss of genetic data during replication (Blackburn, Greider, & Szostak, 2006). During each somatic cell division, however, telomeres shorten by 30-200 base pairs because DNA polymerase is unable to fully replicate the 3' end of the DNA strand (Starkweather et al., 2013). This is referred to as the end replication problem and leads to a decline in TL over time. After approximately 50-70 cell divisions, telomeres reach a critically short length – referred to as the Hayflick Limit – and subsequent processes are activated to signal cellular senescence. Subsequent research has found that degradation of telomeres to this critically short length is associated with a range of adult diseases such as cancer and cardiovascular disease (Haycock et al., 2012; Rode et al., 2015). Thus, telomeres play a protective role in DNA replication and serve as a biological marker of cellular aging and subsequent age-related diseases.

Over the past decade researchers have begun to examine if psychosocial and environmental factors influence TL over time (Oliveira et al., 2016; Price et al., 2011; Starkweather et al., 2013). For example, a recent study by Shalev and colleagues (2013) found that children exposed to two or more forms of violence (i.e., domestic violence, victimization, maltreatment) had shorter telomeres 5 years later compared to those who experienced one or no type of violence exposure. Furthermore, results revealed that maltreatment (i.e., physical or sexual abuse) had the strongest association with TL compared to other forms of violence exposure. Another study by Kiecolt-Glazer and colleagues (2011) found that exposure to two or

more ACEs was inversely associated with TL several decades later, when participants were nearly 70 years old. These results illustrate the negative impact the social environment – particularly ACEs – can have on TL and how these effects can be observed decades later.

### **Protective Factors**

Although much of the literature examining the role of the social environment in predicting TL has focused on risk factors that are associated with accelerated telomere degradation, less attention has been paid to protective factors that prevent rapid telomere attrition – and subsequent poor health outcomes – in response to environmental stress. Briefly, protective factors refer to individual features and/or social experiences that promote positive outcomes *particularly* in situations of risk or adversity (Wright, Masten, & Narayan, 2013). Thus, protective factors typically are conceptualized as moderators in relation to adversity and a particular outcome. From a developmental perspective, protective factors provide one explanation for why certain individuals do not develop negative physical or psychological consequences when exposed to adversity.

Social support is a commonly studied protective factor that consistently has been linked to physical health outcomes throughout the lifespan (Cohen & Wills, 1985; Holt-Lunstad, Smith, & Layton 2010; Schumaker & Czajkowski, 1994). Although definitions vary throughout the literature, social support can be defined as information that one is part of a social network in which they are valued, loved, and cared for by significant others (i.e., family and friends; Taylor, 2010). In the adult literature, the link between social support and health outcomes has been well established (Holt-Lunstad, Smith, & Layton, 2010). For example, a meta-analysis by Holt-Lunstad and colleagues (2010) revealed that – when examining mortality rates – having stronger social relationships was associated with a 50% increased likelihood of survival compared to

those individuals with weaker social relationships. These results elucidate the direct impact that social support can have on physical health; however, recent work has shifted its focus towards examining the mechanisms through which social support indirectly affects physical health outcomes.

Several models have been proposed explaining how social support contributes to various health outcomes, particularly in response to ACEs (e.g., Cohen & Wills, 1985; Taylor, 2010; Uchino, 2006). Of particular interest is Cohen & Wills' (1985) Social Support and the Buffering Hypothesis model (see Figure 2), which suggests that social support may alter an individual's appraisal of a stressful event, mitigating the body's physiological response, and thus decreasing the likelihood of developing negative health consequences associated with the stressful experience. Thus far, research supports the link between social support and alterations in biological processes, such as inflammation, neuroendocrine response, and neural regulation (for reviews, see Taylor, 2010, Uchino, 2006). For example, a study by Eisenberg and colleagues (2007) found that individuals with higher levels of daily social support had lower levels of cortisol reactivity in response to a stress task compared to those with lower levels of daily social support. Building upon this work, researchers have begun to explore how social support affects other biological processes, such as telomere degradation. For example, a recent study by Asok and colleagues (2013) revealed that the effects of early life adversity (i.e., being a member of the Child Welfare System) on TL were significantly diminished among children with mothers engaging in high levels of responsiveness. Together, these results provide ancillary evidence that social support can buffer the biological effects of stress and potentially protect individuals from developing negative health outcomes. Although this work is promising, empirical evidence

examining the moderating role of social support on TL in response to ACEs (particularly CSA) in the adult literature is limited and warrants further examination.

In addition to social support, individual attributes such as personality traits (e.g., pessimism, optimism, neuroticism) have been hypothesized to predict physical health outcomes. Optimism – which can be defined as the tendency to “expect good experiences in the future” (Carver & Scheier, 2001, p. 31) – is one personality trait often linked to better physical health in adulthood (Rasmussen, Scheier, & Greenhouse, 2009; Smith, 2006). As an example, Tindle and colleagues (2009) sampled over 95,000 middle-aged women and tracked their health over an eight-year period. Results from the study revealed that women categorized as optimists (as opposed to pessimists) had a lower risk for coronary heart disease (CHD) and lower mortality rates during the study. In another study, optimism also was linked to a lower incidence of coronary heart disease (CHD; Kubzansky, Sparrow, Vokonas, & Kawachi, 2001). Lastly, a meta-analysis by Rasmussen and colleagues (2009) found that optimism was significantly associated with a variety of health outcomes (e.g., mortality, cardiovascular problems), as well as changes in physiological processes such as inflammation. These results illuminate the role that optimism plays in physical health, and how it can directly affect specific biological processes.

As links between optimism and health have become well documented, Smith (2006) proposed several models for how personality factors may indirectly influence health outcomes through alterations in biological processes. One of these models –the Interactional Stress Moderation Model (see Figure 3) – postulates that when an individual is exposed to stress, personality factors (e.g., optimism) can influence how the person appraises and copes with the situation, affecting biological functioning, which in turn affects health outcomes. However, empirical evidence testing this model is limited (Jobin, Wrosch, & Scheier, 2014; Smith, 2006).

Some research has begun to examine the link between optimism and TL, finding positive associations (e.g., O'Donovan et al., 2009); however, these studies have not examined optimism and TL within an interactional stress moderation framework.

## **Review of the Literature**

**Childhood sexual abuse.** Over the past several decades there has been an increased emphasis on understanding the long-term physical health consequences of CSA (for reviews, see Irish, Kobayashi, & Delahanty, 2009; Wegman & Stetler, 2009). Overall, current meta-analytic reviews have revealed that CSA has small to moderate effects on long-term physical health outcomes, which is similar to the impact of CSA on psychological health. Specifically, Irish and colleagues (2009) found that CSA was associated with an increase in general physical health problems, as well as specific health issues such as pain, obesity, gastrointestinal health, and cardiovascular health. In addition, Wegman and Stetler (2009) found that the largest effect sizes for health outcomes were amongst females – perhaps due to their higher likelihood of being abused and/or reporting abuse – and those studies that used self-report measures of abuse. As the link between CSA and long-term physical health outcomes has been established, researchers have begun to explore the mechanisms through which CSA leads to alterations in physical health over time.

There have been various theories proposed to explain how early life adversity (particularly sexual abuse) impacts physical health outcomes in adulthood (for reviews, see Hulme, 2004; Masten & Cicchetti, 2010; Miller et al., 2009). One theoretical framework in particular – developmental cascades – provides a strong explanation for how the effects of CSA could be observed late into adulthood. Developmental cascades refer to cumulative consequences that occur as a result of many interactions and transactions between an individual and his/her



environment (Masten & Cicchetti, 2010). Thus, the effects of early life adversity may have repercussions lasting many years, which can influence health later in life. A recent study by Jones and colleagues (2016) tested this framework, finding that negative environments at ages 10-12 were associated with related outcomes through ages 30-33 via developmental cascades. For example, a family substance use environment at ages 10-12 was positively associated with youth forming a peer substance use environment at ages 15-18, and developing their own substance use behaviors at age 21. These results support the notion that the social environment can spur cascades that lead to the development of maladaptive outcomes over time; however, the specific mechanisms that drive these cascades are not explained.

More recently, Miller and colleagues (2011) have proposed the Biological Embedding of Childhood Adversity (BECA) Model, which attempts to explain the biological mechanisms linking ACEs to poor long-term health outcomes. Specifically, this model posits that ACEs cause long-term programmed alterations among various biological processes that ultimately contribute to adult disease. Further, these biological alterations can be amplified by certain behavioral proclivities, such as poor social ties and unhealthy lifestyle choices. As an example, a study by Kiecolt-Glaser and colleagues (2011) found that adults exposed to various forms of childhood abuse showed significantly higher levels of interleukin-6 compared to those reporting no childhood abuse; this cytokine plays an integral role in the development of age-related diseases and long-term dysregulation has implications for physical health (Miller et al., 2009). One goal of the current study was to test part of the BECA model with a developmental cascades framework in mind to examine if early life adversity (i.e., sexual abuse) leads to long-term alterations in biological processes (e.g., telomere attrition) that have implications for health outcomes later in life.

**Telomere attrition as a mechanism.** As previously discussed, telomeres are one biological marker that have been linked to both ACEs and adult physical health. Further, they may provide one explanation for how CSA “gets under the skin” and leads to long-term health outcomes. Although numerous studies have examined the link between ACEs and TL, far fewer studies have specifically examined the effect of CSA on TL. As such, the current literature review will focus more broadly on the effects of childhood maltreatment on TL and address gaps in the CSA literature.

There are several studies examining the link between childhood maltreatment and TL, with mixed findings. For example, a study by Tyrka and colleagues (2010) found that healthy adults (aged 18-64) exposed to childhood maltreatment had significantly shorter mean TL compared to individuals experiencing no maltreatment. However, CSA was excluded from analyses because too few individuals endorsed it. Conversely, a study by O’Donovan and colleagues (2011) found an inverse association among childhood trauma (including sexual abuse) and mean TL among adults (~ 30 years old) with PTSD. Lastly, a prospective study by Shalev and colleagues (2013) found that children exposed to physical maltreatment at age 5 had significantly shorter mean TL at age 10 compared to children who reported no exposure to maltreatment. However, maltreatment was assessed via parent reports and CSA was coupled with other forms of physical maltreatment, making it unclear if CSA was driving this association. Altogether, these studies finding positive associations between childhood maltreatment and TL are promising, but studies with thorough self-report measures of CSA among middle-aged adults are warranted to capture more accurate, long-term effects of CSA on TL.

There also are several studies that have failed to find a significant association between childhood maltreatment (including CSA) and TL. For example, a study by Glass and colleagues

(2010) found no significant difference in mean TL between adults who had reported a history of CSA and/or physical abuse and those who reported no abuse. Another study by Kiecolt-Glaser and colleagues (2011) sampled older adults (~ 70 years old), finding that individuals exposed to two or more ACEs had significantly shorter mean TL compared to older adults with no ACEs; however, physical abuse (including CSA) was not independently associated with TL. A study by Surtees and colleagues (2011) did find an association between certain ACEs (e.g., parental alcohol abuse) and mean TL, but no significant association between physical abuse and TL. Lastly, the most recent study to-date by Mason and colleagues (2015) sampled middle-aged females and found a moderate association between physical abuses and mean TL, but no significant association for sexual abuse. Further, this study did have a more thorough measure of CSA, examining the age period for which the abuse occurred (0-10 vs. 11-17) and varying levels of severity (i.e., sexual touching only versus threatening, holding down, or hurting the victim during abuse). Overall, the mixed findings linking CSA (and maltreatment more broadly) to TL depict an equivocal association. Furthermore, these ambiguous findings may be, in part, due to the limited (or lack of) measures regarding CSA. Thus, another goal of the current study was to examine the link between CSA and TL to better understand this association.

**The role of protective factors.** Although the relation between ACEs and TL is often examined within the context of risk, it is necessary to explore and understand the role of resilience in this association. For example, the developmental cascades model suggests that factors in one domain (e.g., positive family environment) can have effects in another (e.g., peer substance use environment). One such factor that has been hypothesized to influence biological processes and physical health is social support. Specifically, social support has been proposed to

alter a person's response (e.g., appraisal) to stressful experiences, which attenuates the biological impact of the stressor and limits the subsequent physical health effects (Cohen & Wills, 1985).

Only one study to-date has examined the protective effects of social support on TL (Asok et al., 2013). Specifically, the authors found that maternal responsiveness – a type of social support within families – mitigated the impact of childhood adversity (defined as being in the Child Welfare System) on TL in young children. Aside from this work, researchers have focused primarily on examining the direct link between social support and TL. For example, a study by Carroll and colleagues (2013) found that social support among older adults (65-84 years old) was positively associated with TL. In addition to this work, Beach and colleagues (2014) found an inverse relationship between unsupportive parenting and TL in adolescents. Further, an intervention study using this sample found that adolescents whose parents participated in the intervention and reported increases in levels of warmth and emotional support had significantly longer telomeres five years later compared to those adolescents in a control group (Brody et al., 2014). Several other studies also have found significant associations between social support and TL (Barger & Cribbet, 2016; Diaz et al., 2010; Uchino et al., 2012; Zalli et al., 2014), but research on this link is still in its infancy.

Although much of the research examining social support and TL has focused on examining direct associations, evidence suggest that social support serves as a protective factor in the context of risk (e.g., Holt-Lunstad et al., 2010; Taylor, 2010), and further examination of social support as a protective factor in relation to TL is needed. Thus, another goal of the current study was to examine if social support buffers the effects of CSA on TL in a sample of adults.

Optimism is another factor that has been hypothesized to serve a protective role in the association between ACEs, biological processes, and subsequent health outcomes. Specifically,

certain aspects of personality (e.g., optimism) have been hypothesized to influence an individual's psychological appraisal of stress and coping behaviors, which have a direct influence on an individual's physiological response to stress and subsequent health outcomes (Smith, 2006). Recently, researchers have begun to explore how personality factors relate to TL. For example, a recent study by Schutte and colleagues (2016) examined the association between various psychological characteristics and telomere length in middle-aged women. Results from the study revealed that positive psychological characteristics explained a significant amount of variance in TL ( $R = .40$ ). Looking at independent characteristics, the authors found that optimism was significantly associated with TL ( $r = .30$ ), even after controlling for age, gender, and other psychological traits. Similarly, Zalli and colleagues (2014) found that older men with shorter telomeres and high telomerase activity – a combination that is hypothesized to be indicative of a stressed system – reported lower levels of optimism compared to the rest of the sample. These studies provide preliminary evidence that optimism can impact TL; however, not all studies have found significant associations between optimism and TL (Ikeda et al., 2014; O'Donovan et al., 2009; Rius-Ottenheim et al., 2012), and this association has yet to be tested in the context of CSA.

### **Statement of the Problem**

Although CSA has been linked to poor physical health outcomes later in life (Irish et al., 2009; Wegman & Stetler, 2009), the mechanisms linking CSA and adult physical health are still under debate. Telomere attrition is one biological process that may serve as a viable mechanism linking CSA to physical health outcomes; however, literature examining the association between CSA and TL is limited. In addition, associations between ACEs and TL often have been examined within the context of risk. Theory and empirical evidence, however, suggest that

certain factors can protect against rapid telomere attrition, ultimately preventing poor health outcomes (e.g., Asok et al., 2013; Schutte et al., 2016). Thus, the role of CSA in predicting TL is unclear, and examination of protective factors in this association is warranted.

### **Present Study**

The present study contributes to the literature on the long-term effects of ACEs by examining one potential mechanism through which ACEs can impact physical health later in life. Child sexual abuse is a severe (and sometimes chronic) ACE that has been linked to poor health later in life (Irish et al., 2009; Wegman & Stetler, 2009); however, the mechanisms through which these effects occur are not well understood. Over the past decade, however, telomeres have become a biological marker of interest, as their progressive shortening has been linked to both ACEs and adult health outcomes (Oliveira et al., 2016; Rode et al., 2015). Several studies have examined the association between CSA and TL in adults; however, findings are mixed and measures of CSA are often blunted, perhaps leading to this equivocal association. Thus, the present study first examines the association between CSA and TL using a sample of female monozygotic (MZ) twins who are discordant for CSA. The advantage of utilizing a discordant MZ twin design lies in the ability to control for genetic and shared environmental influences between twins, which aids in inferring causality within a cross-sectional design.

Another contribution of the present study is the examination of protective factors on the association between CSA and TL. To date, many of the studies examining the impact of ACEs on TL have examined this association from a perspective of risk (Oliveira et al., 2016); however, theory and empirical evidence suggests that social support and optimism are linked to health outcomes later in life and may serve as protective factors by mitigating the biological impact of ACEs (Cohen & Wills, 1985; Smith, 2006). However, this has yet to be explored in the CSA

literature. Thus, another goal of the current study was to examine the moderating effects of social support and optimism on the association between CSA and TL in adults. Results of these analyses serve to inform our understanding the complex relationship between CSA, TL, and protective factors, while also informing prevention and intervention research efforts.

### **Statement of the Hypotheses**

**Hypothesis 1.** Individuals exposed to any CSA will have significantly shorter mean TL compared to individuals who have no history of CSA.

**Hypothesis 2.** Social support will have a significant moderating effect on the association between CSA and TL. Specifically, females exposed to CSA who have higher levels of social support will have longer mean TL compared to females exposed to CSA with lower levels of social support.

**Hypothesis 3.** Optimism will have a significant moderating effect on the association between CSA and TL. Specifically, females exposed to CSA who have higher levels of optimism will have longer mean TL compared to females exposed to CSA with lower levels of optimism.

### **Methods**

#### **Participants**

The present sample consists of a subset of female-female (FF) monozygotic (MZ) twins ( $N = 99$ ) who participated in the population-based Virginia Adult Twin Study for Psychiatric and Substance Use Disorders (VATSPSUD; for more information on this study and its design, see Kendler & Prescott, 2006). Participants in the current study were drawn from the FF Twin Study, which began data collection in January of 1987. The FF Twin Study consisted of individuals whom were born between 1934-1974 and ranged in age from 18-54 years, with the average age of participants being 29.3 years ( $SD = 7.7$ ) at Wave 1. All participants identified as Caucasian,

median household income was \$35,000-40,000 and average years of education was 13.5 (i.e., some college). The analytic sample consisted of females ranging in age from 19-48 ( $M_{\text{age}} = 30.5$ ,  $SD = 7.8$ ) with an average of 13.9 years ( $SD = 2.0$ ) of education, and median income of \$30,000-35,000 at Time 1. There were 22 pairs discordant for CSA ( $n = 44$ ), 13 pairs concordant for CSA ( $n=26$ ), and 29 singletons (i.e., a twin whose co-twin did not participate in the study), 23 of which were exposed to CSA.

## **Measures**

**Childhood sexual abuse.** Sexual abuse during childhood was assessed via a self-report questionnaire at Wave 4, which was approximately eight years after Wave 1. The questionnaire used was an adapted version of a previously developed measure by Mullen and colleagues (1993). The measure consists of a single item stem asking, “Before you were 16, did any adult, or any other person older than yourself, involve you in any unwanted incidents like...” and participants chose from a list of six different forms of sexual abuse. Response options ranged on a scale from 0 (never) to 2 (more than once), and could be further broken down into three categories of abuse: non-genital (3 items), genital without intercourse (2 items), and intercourse (1 item). A binary version of this variable was created where endorsement of any form of CSA was coded as a “1” and no endorsement as “0.” Lastly, a variable was created to assess severity of abuse (low, medium, high), based on participants reporting non-genital abuse (low severity), genital without intercourse (medium severity) and intercourse (high severity).

**Social support.** Social support was assessed at Wave 1 using a subset of self-report items from an original measure created for the VATSPSUD study (Kendler & Prescott, 2006). This subscale consists of 10 items that assess the quality of support obtained from friends (5 items) and relatives (5 items). A sample item from this measure is, “When you are in contact with



friends, how often do they make that you feel like they care about you?” Responses ranged from 1 (often) to 4 (never). Items responses were recoded so that higher scores indicate higher levels of support. Scores across support groups (i.e., friends and relatives) were summed to create a total score for social support. Reliability for these items among the analytic sample was marginal ( $\alpha = .69$ ).

**Optimism.** Optimism was assessed at Wave 1 using a subset of items from the Life Orientation Test (LOT; Scheier & Carver, 1985). This self-report measure has previously demonstrated good reliability and validity. Optimism was assessed via five items that examined a participant’s outlook on life (e.g., “I always look on the bright side of things”). Responses ranged from 1 (strongly agree) to 4 (strongly disagree). Item responses were recoded so that higher scores indicate higher levels of optimism. Scores were summed to create a total score for optimism. Reliability for these items among the analytic sample was adequate ( $\alpha = .75$ ).

**Telomere length.** Telomere data collection began approximately 10 years after Wave 4, which will be referred to as Wave 5. Prior to the current study, a mean T/S ratio value was calculated for each individual in the current sample by averaging across three individual measurements of telomere length that were obtained for each participant (described in detail below). A research technician examined these measurements and if one of the three measurements varied significantly from the other two measurements, it was removed from the mean calculation (i.e., the mean T/S ratio consisted of only two measurement values). Although this method was rather arbitrary, it was relatively clear when a telomere length measurement may have been incorrect. Once final T/S ratio values were obtained, a histogram was created to assess for potential outliers.

***DNA isolation.*** Genomic DNA was extracted from whole blood using a Puregene DNA Isolation Kit (Qiagen) according to the manufacturer's protocol. After extraction, the DNA was quantified, evaluated to ensure it was not degraded, and stored at -80°C until used for telomere assessments. DNA stocks were diluted to approximately 20 ng into pure water prior to setting up qPCR runs.

***Monochrome multiplex qPCR (MMqPCR).*** The relative genomic telomere length for the co-twins was measured using a monochrome multiplex real-time quantitative PCR (MMqPCR) technique, as previously described (Cawthon, 2009). Briefly, in this method relative telomere length was quantified as a ratio of the number of copies of the telomere repeat sequences compared to the number of copies of a single housekeeping gene (human albumin), with this number being derived from a standard curve. This approach eliminates potential DNA measuring errors that could arise if the telomere and reference DNA were evaluated from aliquots into different wells. Also, to reduce experimental variation, also called "batch effects", specimens from co-twins were placed on the same multi-well plates.

All co-twin specimens were run in triplicate (3 different wells per specimen). In addition to the co-twin specimens, each plate contained standard DNA (a "cocktail" of DNA specimens derived from 11 healthy control women of comparable age to the co-twins), which was also run in triplicate. Each plate also contained DNA from three "control" specimens (run in triplicate). The DNA from the controls was derived from: (1) MCF7 cells [known to have short telomeres]; (2) HeLa cells [known to have long telomeres], and (3) JMS cells [known to have moderate telomere lengths]. The telomere (T) to standard (S) gene ratio values for these control specimens were assessed for each plate to ensure that each control showed small (MCF7), large (HeLa) or

moderate (JMS) values before including the co-twin values from that batch in the dataset for analysis.

As described by Cawthon (2009), the primer sequences used to amplify the telomere sequences were:

telg: AACTAAGGTTTGGGTTTGGGTTTGGGTTTGGGTTAGTGT

telc: TGTTAGGTATCCCTATCCCTATCCCTATCCCTATCCCTAACA

The standard “housekeeping” gene used for this assay was albumin (which showed optimal amplification results in validation studies). The primers used to amplify the albumin sequences were:

albu: CGGCGGCGGGCGGCGCGGGCTGGGCGGaaatgctgcacagaatccttg

albd: GCCCGGCCCGCCGCGCCCGTCCCGCCGaaaagcatggtcgctgtt

For all specimens (co-twin, standard, and controls) each well contained a 5 µL aliquot of DNA (diluted to 4 ng/µL), 15 µL of a SYBR Green “Mastermix”, and primers for the telomere and albumin sequences. Plates were run on a BioRad CFX96 with the following cycling parameters: 95°C for 5 minutes; 2 cycles of 94°C for 15 seconds, 49°C for 15 seconds; 49 cycles of 15 seconds at 94°C, 15 seconds at 62°C, 15 seconds at 83°C, 15 seconds at 60°C with signal acquisition, 15 seconds at 94°C, 20 seconds at 85°C with signal acquisition. The 60°C reads provided the Ct values for the amplification of the telomere template (in early cycles when the albumin signal is still at baseline). The 85°C reads provided the Ct values for the amplification of albumin template at which time the telomere template is fully melted.

A 5-point standard curve was derived from triplicate measures of the “cocktail” mix of DNA from the 11 healthy control women and was prepared in dilutions ranging from 1.235 to 100 ng.

The BioRad CFX Manager software version 3.0 was used to calculate the value of T (telomere) and S (albumin single copy gene) using the standard curve.

### **Covariates**

Age at time of telomere data collection (i.e., Time 5), body mass index (BMI), smoking status, education level, lifetime diagnosis of Major Depressive Disorder (MDD), and lifetime diagnosis of Generalized Anxiety Disorder (GAD) were identified as relevant covariates due to their theoretical and empirical associations with TL (Starkweather et al., 2013). Height (in meters) and weight (in kilograms) were collected from study participants at Time 1, and BMI was calculated using a standard formula:  $[(\text{weight}) \div (\text{height}^2)]$ . Smoking status (i.e., “Are you currently smoking regularly?”) was collected at Time 4 via a yes/no self-report item. Education level was assessed at Time 4 via a self-report item asking participants to report the number of years of education they had completed prior to the interview. Responses ranged on a scale from 0 (no schooling) to 20+ (doctorate). Lastly, lifetime diagnosis of MDD and GAD (i.e., yes/no) was assessed at Wave 4 via a slightly adapted version of the Structured Clinical Interview for DSM-III-R diagnoses (Spitzer, Williams, & Gibbon, 1987). A more detailed description of assessment can be found elsewhere (cf, Kendler & Prescott, 2006), but test-retest reliability for lifetime MDD and GAD diagnoses was good (i.e.,  $\kappa > .70$ ). In order to preserve power given the small sample size, associations were tested between each covariate and TL, and only those covariates that had a significant association with TL were considered for use in subsequent analyses. Age was the only exception to this rule; since telomeres are a biological marker of aging, best practice dictates that this covariate be included in all analyses.

### **Procedure**

Participants in the current study were originally identified and recruited for VATSPSUD through the Virginia Twin Registry (VTR) by matching birth dates to state records. The original recruitment targeted FF twin pairs born between 1934 and 1970. The registry identified 1,176 twin pairs (2,352 individuals) who were then sent questionnaires in order to participate in the study. Of these 2,352 women, 2,164 individuals (1,033 complete twin pairs) completed and returned at least one questionnaire and were included in the study at Wave 1. There was at least a one-year break between waves of data collection, with an average interval between Wave 1 and Wave 2 of 17.3 months ( $SD = 3.8$ , range = 12-49), 45.0 months ( $SD = 4.0$ , range=30-53) between Wave 2 and Wave 3, and 36.1 months ( $SD = 8.8$ , range = 17-58) between Wave 3 and Wave 4. Approximately 10 years after Wave 4, participants who had responded to questions regarding CSA history were invited to complete a health history questionnaire and provide blood samples. After providing their informed consent, blood samples were obtained by a health care provider of the participant's choosing and shipped to a cytogenetic laboratory at room temperature per standard procedures. Telomere length data were obtained from these samples (see description above), and mean T/S ratio values were calculated for each participant in the sample.

### **Analytic Strategy**

Preliminary analyses for the current study were conducted using SPSS version 22 (IBM, 2013) and study hypotheses were tested using RStudio version 1.0.136 (RStudio Team, 2016). Before beginning study analyses, data were cleaned and checked. Specifically, social support and optimism items were recoded so that higher scores indicated higher levels of each construct. Descriptive data also were obtained to examine the distribution of each variable. Histograms were created for each study variable to assess for potential outliers (i.e., a value  $\pm 2 SD$  from

the mean). Lastly, reliability was calculated for the optimism and social support scales for the analytic sample. Once data cleaning and checking was complete, bivariate correlations – or linear mixed effects models when appropriate – were run for each covariate and TL for all study participants.

Hypothesis 1 was first tested via a paired-samples *t*-test using only discordant MZ twin pairs; however, to increase sample size (and subsequent power) while still taking in to account dependence of observations (i.e., twins nested within pairs), this hypothesis also was tested via a linear mixed effects model – allowing for a random effect of family membership – with maximum likelihood estimation – using the “lme4” package (Bates, Maechler, Bolker, & Walker, 2015) – in RStudio (RStudio Team, 2016). Exposure to any child sexual abuse (yes/no) was the independent variable and mean TL was the dependent variable. Age was included in the model as a covariate. These tests were one-tailed with a *p*-value of .05.

Hypothesis 2 was tested via linear mixed effects models – allowing for a random effect of family membership – with maximum likelihood estimation – using the “lme4” package (Bates et al., 2015), controlling for age in all models (see Figure 4). The independent variable was exposure to any CSA; the moderator variable was social support; and the dependent variable was mean TL. Before running the analyses for hypothesis 2, any continuous predictor or moderating variables were mean-centered (i.e., social support, age). Next, an interaction term was created, whereby the binary CSA variable was multiplied by the mean-centered social support variable. All variables were entered into the first model simultaneously for analysis. A second, reduced model was then tested without the interaction term, and a likelihood ratio test was conducted to assess if there was a significant decrease in model fit when excluding the interaction term. Lastly, if a statistically significant interaction emerged, mean TL was plotted for both CSA

exposed and CSA non-exposed twins, at low (1 *SD* below the mean), medium (mean), and high (1 *SD* above the mean) levels of the moderator, and a simple slopes analysis was conducted.

In addition to the analyses above, two identical models were run with the CSA severity variable as the main predictor – including all study participants –, where 0 = unexposed, 1 = non-genital exposure, 2 = genital exposure, and 3 = intercourse. Although CSA exposure often is tested in the literature as a binary (i.e., yes/no) variable, exposure to CSA can potentially be viewed as an ordinal or continuous variable. Thus, this version of the variable also was used as a main predictor to further elucidate the effects of CSA on mean TL. Lastly, two more identical models were tested (with CSA severity as the main predictor) – excluding the unexposed individuals ( $n = 28$ ). If CSA severity is to be treated as a continuous variable, then the distance between each “point” is assumed to be equal. As such, to include individuals unexposed to abuse may bias the findings since the difference between no abuse and sexual abuse is significant. If any significant interactions emerged in these models, interaction plots were created assessing mean TL as a function of CSA severity at high, medium, and low levels of the moderator, and a simple slopes analysis was conducted. In all, 6 models were tested for the second hypothesis.

Hypothesis 3 was tested via linear mixed effects models – allowing for a random effect of family membership – with maximum likelihood estimation – using the “lme4” package (Bates et al., 2015), controlling for age in all models (see Figure 4). The independent variable was exposure to any CSA; the moderator variable was optimism; and the dependent variable was mean TL. Before running the analyses for hypothesis 3, any continuous predictor or moderating variables were mean-centered (i.e., optimism, age). Next, an interaction term was created, whereby the binary CSA variable was multiplied by the mean-centered optimism variable. All variables were entered into the first model simultaneously for analysis. A reduced model was

then tested without the interaction term, and a likelihood ratio test was conducted to assess if there was a significant decrease in model fit when excluding the interaction term. Lastly, if a statistically significant interaction emerged, mean TL was plotted for both CSA exposed and CSA non-exposed twins, at low (1 *SD* below the mean), medium (mean), and high (1 *SD* above the mean) levels of the moderator, and a simple slopes analysis was conducted.

In addition to the analyses above, two identical models were run with the CSA severity variable (including all participants), where 0 = unexposed, 1 = non-genital exposure, 2 = genital exposure, and 3 = intercourse, for reasons explained in hypothesis 2. Lastly, two more identical models were tested, excluding the unexposed individuals in order to get a clearer picture of the effects of optimism on the relation between CSA severity and mean TL. If any significant interactions emerged, interaction plots were created assessing mean TL as a function of CSA severity at high, medium, and low levels of the moderator, and a simple slopes analysis was conducted. In all, 6 models were tested for the third hypothesis.

## **Results**

### **Missing Data**

Although all study participants had full data for CSA exposure (or non-exposure), telomere length, and age, several individuals had missing data for type of CSA exposure, social support, and optimism. Although it was not reasonable to impute responses for CSA exposure type, scores were imputed for some cases missing data on the social support and optimism scales in order to preserve as many cases as possible given the limited sample size. Specifically, if an individual answered at least one item on either scale, a sum was computed – based on the mean of completed items – for that individual using all available responses for that scale. This procedure was only used for two individuals' social support scale score. Cases missing all data



for the social support ( $n = 8$ ) or optimism ( $n = 12$ ) scales did not receive a total scale score and were thus excluded from their respective analyses.

### **Descriptive Statistics and Correlations between Study Variables**

Table 1 provides descriptive data on non-CSA study variables. There were no issues with skewness or kurtosis in relation to the social support or optimism scales. Data for telomere measurements were kurtotic due to two cases that had unusually high values for their T/S ratio. Raw telomere data for these cases were reviewed and one value was excluded for these cases when telomere measurements were run in triplicate (i.e., only two values were used to create the mean T/S ratio). After conferring with the individual who collected the raw telomere data, it was determined that these two scores were inaccurate and were thus removed from all analyses. All descriptive data herein refer to the analytic sample excluding the two mean TL outliers ( $N = 97$ ).

Table 1

#### *Descriptive Statistics for Non-CSA Study Variables*

Variable	N	Mean (SD)	Range	Skewness	Kurtosis
Mean T/S Ratio	97	1.33 (.40)	.48 – 2.14	.04	-.96
Age	97	52.74 (8.55)	35 - 70	.17	-.65
Social Support	90	33.33 (3.56)	22 - 40	-.30	-.01
Optimism	86	15.07 (2.29)	8 - 20	-.57	.45
Education	97	14.67 (2.14)	12 - 20	.18	-1.08
BMI	90	23.16 (4.76)	17.26 - 40.44	1.60	2.39
Smoking Regularly*	46	16 (34.8%)	-	-	-
Lifetime MDD*	96	43 (44.8%)	-	-	-
Lifetime GAD*	97	17 (17.5%)	-	-	-

*Note.* Mean T/S ratio reflects telomere length measurement. All values reflect data excluding outliers ( $n = 2$ ) for mean T/S ratio. Age reflects age at time of telomere data collection. Range reflects actual range of the data in the study. BMI = Body Mass Index. Asterisk denotes a binary variable (0/1); values in the “Mean (SD)” column reflect the number (and percentage) of participants endorsing that item.

Bivariate correlations between continuous predictors and mean TL revealed that there were no significant associations between social support and optimism ( $r = -.03, p > .05$ ), social support and mean TL ( $r = -.10, p > .05$ ), or optimism and mean TL ( $r = .17, p > .05$ ). When testing associations between a nominal variable (i.e., CSA exposure) and mean TL, social support, and optimism, it was necessary to account for dependent observations in the data (i.e., twins nested within pairs), so linear mixed effects models were employed to test these associations. Results from these models revealed that mean TL was not associated with CSA exposure ( $\beta = .01, p > .05$ ), nor was CSA exposure associated with social support ( $\beta = -.87, p > .05$ ). However, CSA was significantly associated with optimism ( $\beta = -1.31, p = .01$ ), such that individuals exposed to CSA had lower levels of optimism than those individuals without a history of CSA.

Descriptive data also were obtained on the CSA variables. Sixty-nine individuals in the current study reported experiencing CSA, with average age of first abuse being at 10.5 years of age ( $SD = 3.8$ ; range = 3-16 years). Sixteen individuals reported being exposed to non-genital forms of abuse (e.g., kissing or hugging in a sexual way), 29 individuals reported being exposed to abuse involving genitalia (e.g., touching or fondling of private parts), and 24 individuals reported being exposed to abuse involving intercourse (i.e., attempting or having sexual intercourse). Table 2 presents information on the type of response individuals received if they told someone about the abuse, as well as the type of support – or lack thereof – that each abused participant received after revealing their abuse to another person. A majority of the sample (42.2%) did not reveal their abuse to anyone; however, of those individuals that did reveal their abuse to another person, a large portion were believed and supported (19.2%). Similarly, the

majority of individuals who revealed their abuse received a positive response (13.1%) from the person they revealed their abuse to.

Table 2

*Proportion of Responses from Individuals Exposed to CSA*

Support Items	
Believed and supported ( $N = 62$ )	27.4%
Believed but not supported ( $N = 61$ )	11.5%
Not believed ( $N = 60$ )	3.3%
Blamed or punished ( $N = 60$ )	3.3%
I told no one ( $N = 62$ )	67.7%
Response Items	
Positive Response ( $N = 69$ )	17.4%
Mostly Positive Response ( $N = 69$ )	4.3%
Neutral Response ( $N = 69$ )	5.8%
Mostly Negative Response ( $N = 69$ )	4.3%
Negative Response ( $N = 69$ )	2.9%
Never told anyone ( $N = 69$ )	52.2%

*Note.* Support items stem was, “I told someone and was...”

**Covariates.** In order to establish covariates to be used in subsequent analyses, bivariate correlations were run between continuous covariates and TL. There were no statistically significant correlations between TL and age ( $r = -.09, p > .05$ ), BMI ( $r = -.11, p > .05$ ), or education level ( $r = .03, p > .05$ ); however, because telomeres are a biological marker of aging, age was still included as a covariate in subsequent analyses. Linear mixed effects models were employed to examine if there were significant associations between mean TL and nominal covariates. Results from these analyses revealed that TL was not significantly associated with lifetime MDD ( $\beta = .11, p > .05$ ), lifetime GAD ( $\beta = .10, p > .05$ ), or smoking status ( $\beta = -.07, p >$

.05). Although age at Time 5 was the only covariate included in the final analyses, lifetime MDD was considered for use as a covariate because it is possible that – as a consequence of CSA exposure – MDD could confound the association between CSA and mean TL. However, MDD was not associated with mean TL, and models including MDD (results not reported) revealed the same findings as models excluding this variable, so it was deemed appropriate to exclude lifetime MDD from the final analyses.

### **Hypothesis 1**

First, a paired-samples *t*-test was conducted with only discordant MZ twin pairs ( $n = 22$ ) to test hypothesis 1, that females exposed to any CSA would have shorter mean TL compared to females with no history of abuse. Results from this test indicated no association between CSA exposure and mean TL,  $t(21) = .08, p > .05$ . Next, in an effort to increase sample size while accounting for dependence of observations in the sample, a linear mixed effects model was conducted to test hypothesis 1 using all available observations ( $N = 97$ ). P-values were obtained for all coefficients using the “lmerTest” package (Kuznetsova, Brockhoff, & Christensen, 2016). Results again revealed that there was no evidence for an association between CSA status ( $\beta = .02, p > .05$ ) or age ( $\beta = -.01, p > .05$ ) and mean TL. In sum, there is no evidence to suggest that exposure to CSA impacts mean TL, controlling for known covariates.

### **Hypothesis 2**

Next, hypothesis 2 was tested, which stated that social support would moderate the association between CSA exposure and mean TL. Model results for the binary CSA variable (i.e., no CSA versus any CSA) are presented first. As can be seen in Table 3, the full model including the interaction term revealed no significant effects of any predictor on mean TL. The model excluding the interaction term did not reveal any significant effects either, and the

likelihood ratio test (LRT) comparing these two models revealed no significant decrease in model fit by excluding the interaction term,  $\chi^2(1) = 2.36, p > .05$ . Overall, there was no evidence to suggest that exposure to CSA or social support directly affected mean TL, nor was there evidence to suggest that social support moderated the association between CSA exposure and mean TL.

Table 3

*Fixed Effects from the Full Model of Hypothesis 2 Using the Binary CSA Variable*

Variables	$\beta$	<i>p</i> -value
CSA	-.01	.86
Age	-.01	.34
Social Support	-.03	.12
CSA x Social Support	.04	.12

*Note.* Model fit information for the full model: AIC = 93.1; BIC = 110.6; -2 Log Likelihood = 79.2. Model fit information for the reduced model: AIC = 93.5; BIC = 108.5; -2 Log Likelihood = 81.6.

Next, the same two models were run, treating CSA as a continuous variable, including unexposed individuals in the analyses (i.e., 0 = unexposed, 1 = non-genital, 2 = genital, 3 = intercourse). Results from the full model including the interaction term revealed no significant effects of any predictors in the model (see Table 4). Further, the LRT revealed no significant decrease in model fit by excluding the interaction term from the model,  $\chi^2(1) = .04, p > .05$ . In sum, there was no evidence to suggest that CSA severity or social support directly affected mean TL, nor was there evidence to suggest that social support moderated the association between CSA severity and mean TL.

Table 4

*Fixed Effects from the Full Model of Hypothesis 2 Using CSA Severity (all participants)*

Variables	$\beta$	<i>p</i> -value
CSA	.001	.98

Age	-.01	.39
Social Support	-.01	.63
CSA x Social Support	.001	.84

*Note.* Model fit information for the full model: AIC = 95.5; BIC = 113.0; -2 Log Likelihood = 81.4. Model fit information for the reduced model: AIC = 93.5; BIC = 108.5; -2 Log Likelihood = 81.6.

Lastly, in order to get a clearer picture of the effects of CSA severity on mean TL, analyses were re-run to examine if social support moderated the association between CSA severity and mean TL, excluding unexposed individuals (i.e., 0 = non-genital, 1 = genital, 2 = intercourse). Results from the full model including the interaction term revealed a significant main effect of social support on mean TL, such that increases in social support were associated with increases in mean TL (see Table 5). In addition, the interaction term was significant in the model. The reduced model excluding the interaction term revealed no significant effects of any predictors, but the LRT revealed that exclusion of the interaction term led to a significant decrease in model fit,  $\chi^2(1) = 6.71, p = .01$ . Since a significant interaction emerged, the interaction was plotted at low (1 *SD* below the mean), medium (mean), and high (1 *SD* above the mean) levels of the moderator. In addition, simple slopes analyses were conducted, revealing a significant slope only for low levels of social support ( $p = .03$ ). As can be seen in Figure 5, social support only appeared to impact mean TL for women exposed to non-genital abuse, with higher levels of support associated with longer mean TL. For women exposed to genital abuse or intercourse, social support did not appear to be associated with mean TL.

Table 5

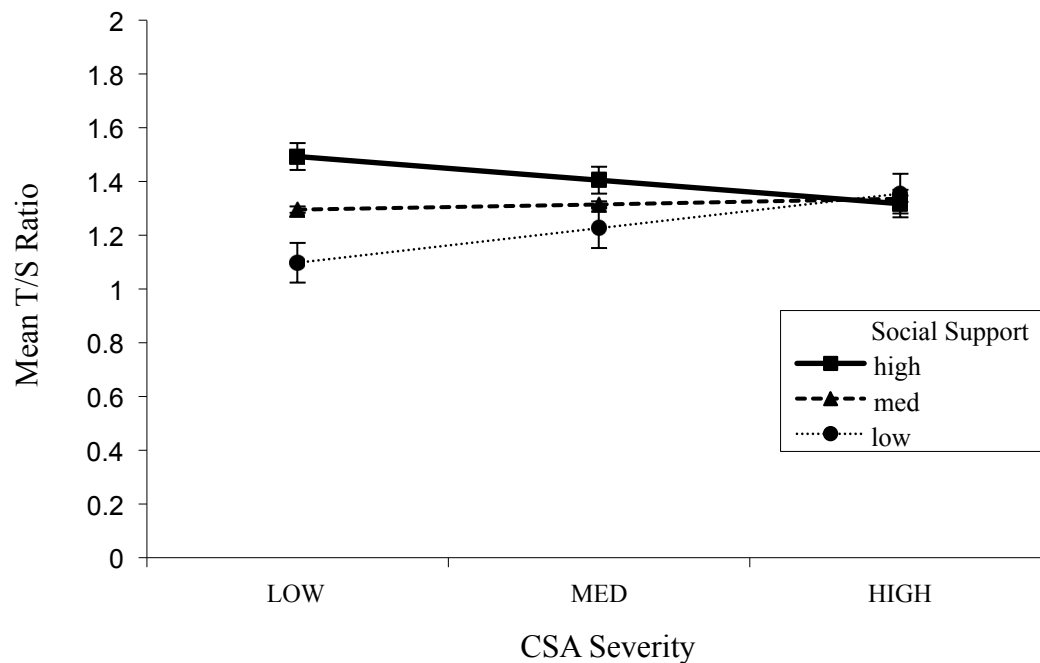
*Fixed Effects from the Full Model of Hypothesis 2 Using CSA Severity (exposed participants only)*

Variables	$\beta$	<i>p</i> -value
CSA	.03	.61

Age	-.01	.36
Social Support	.07	.01*
CSA x Social Support	-.04	.01*

*Note.* Model fit information for the full model: AIC = 63.8; BIC = 78.9; -2 Log Likelihood = 49.8. Model fit information for the reduced model: AIC = 68.5; BIC = 81.5; -2 Log Likelihood = 56.6. \*  $p < .05$ .

**Figure 5.** Interaction between CSA Severity and Social Support Predicting Telomere Length



*Note.* Along the x-axis, low = non-genital abuse, medium = genital abuse, high = intercourse. Simple slopes analyses revealed that the only the slope for low levels of social support was significant ( $p = .03$ ).

### Hypothesis 3

Lastly, hypothesis 3 was tested, which stated that optimism would moderate the association between CSA and mean TL. Model results for the binary CSA variable are presented first. As can be seen in Table 6, the full model including the interaction term revealed no significant effects of any predictor on mean TL. The model excluding the interaction term did not reveal any significant findings either, and the likelihood ratio test (LRT) comparing these

two models revealed no significant decrease in model fit by excluding the interaction term,  $\chi^2 (1) = .01, p > .05$ . Overall, there was no evidence to suggest that exposure to CSA or optimism directly affected mean TL, nor was there evidence to suggest that optimism moderated the association between CSA exposure and mean TL.

Table 6

*Fixed Effects from the Full Model of Hypothesis 3 Using the Binary CSA Variable*

Variables	$\beta$	p-value
CSA	.06	.46
Age	-.01	.29
Optimism	.03	.47
CSA x Optimism	-.004	.91

*Note.* Model fit information for the full model: AIC = 80.4; BIC = 97.6; -2 Log Likelihood = 66.4. Model fit information for the reduced model: AIC = 78.5; BIC = 93.2; -2 Log Likelihood = 66.4.

Next, the same two models were run, treating CSA as a continuous variable, including unexposed individuals in the analyses (i.e., 0 = unexposed, 1 = non-genital, 2 = genital, 3 = intercourse). Results from the full model including the interaction term revealed no significant effects of any predictor on mean TL (see Table 7). Further, the LRT revealed no significant decrease in model fit by excluding the interaction term from the model,  $\chi^2 (1) = .07, p > .05$ . In sum, there was no evidence to suggest that CSA severity or optimism directly affected mean TL, nor was there evidence to suggest that optimism moderated the association between CSA severity and mean TL.

Table 7

*Fixed Effects from the Full Model of Hypothesis 3 Using CSA Severity (all participants)*

Variables	$\beta$	p-value
CSA	.02	.47
Age	-.01	.33



Optimism	.01	.35
CSA x Optimism	-.003	.80

*Note.* Model fit information for the full model: AIC = 80.5; BIC = 97.6; -2 Log Likelihood = 66.4. Model fit information for the reduced model: AIC = 78.5; BIC = 93.2; -2 Log Likelihood = 66.6.

Lastly, in order to get a clearer picture of the effects of CSA severity on mean TL, analyses were re-run for hypothesis 3, excluding unexposed individuals (i.e., 0 = non-genital, 1 = genital, 2 = intercourse). Results from the full model including the interaction term revealed no significant effects of any predictors on mean TL (see Table 8). The reduced model did not reveal any significant effects. Lastly, the LRT revealed that exclusion of the interaction term did not lead to a significant decrease in model fit,  $\chi^2(1) = .07, p > .05$ . In sum, there was no evidence to suggest that CSA severity or optimism directly affected mean TL, nor was there evidence to suggest that optimism moderated the association between CSA severity and mean TL.

Table 8

*Fixed Effects from the Full Model of Hypothesis 3 Using CSA Severity (exposed participants only)*

Variables	$\beta$	<i>p</i> -value
CSA	.03	.72
Age	-.01	.36
Optimism	.01	.46
CSA x Optimism	-.001	.79

*Note.* Model fit information for the full model: AIC = 67.7; BIC = 82.5; -2 Log Likelihood = 53.8. Model fit information for the reduced model: AIC = 65.8; BIC = 78.4; -2 Log Likelihood = 53.8.

### Post-Hoc Analyses

In an effort to clarify the contradictory findings that social support had a buffering effect on the association between CSA severity and mean TL, but only at low levels of severity, several

analyses were conducted. First, since the reliability for the social support scale was low ( $\alpha = .69$ ) I explored the reliability of each set of social support items separately (i.e., family and friends). The reliability for items assessing family members' social support was adequate ( $\alpha = .77$ ), but the reliability for items assessing friends' social support was low ( $\alpha = .59$ ). In order to see if combining the social support scales biased the results, analyses were re-run for the model that revealed a significant interaction between social support and CSA severity using social support from family and social support from friends, separately. The model assessing friends' social support revealed no significant findings, and the model assessing family social support yielded findings with marginal significance for the main effect of social support ( $\beta = .06, p = .06$ ) and the interaction term ( $\beta = -.04, p = .06$ ). These findings suggest that the original results may be biased by inclusion of the friends' social support items, but a meaningful relation between CSA, social support, and mean TL does seem to exist.

## **Discussion**

Sexual abuse during childhood is a traumatic event that has long-term psychological and physical health implications for its victims (Browne & Finkelhor, 1986; Irish, Kobayashi, Douglas, & Delahanty, 2009; Polusny & Follette, 1995; Wegman & Stetler, 2009). Although these long-term outcomes have been well documented, the mechanisms linking CSA to long-term health problems is still under debate. Recent theoretical and empirical inquiries suggest that telomere attrition may serve as one mediating mechanism between exposure to early life stress and later health outcomes (Oliveira et al., 2016; Price et al., 2011). However, a majority of this work has examined the role of telomeres in response to social stress from a risk point of view, ignoring the role of protective factors in mitigating the biological impact of early life stress. Two factors – social support and optimism – consistently have been linked to long-term health

outcomes (e.g., Holt-Lunstad et al., 2010; Rasmussen et al., 2009) as well as alterations in various biological processes that contribute to these health outcomes (e.g., Smith, 2006; Taylor, 2010). The goal of the current study was to examine the effects of a severe early life stressor (CSA) on telomere length in adults, and to examine the moderating role of social support and optimism, to gain a better understanding of the characteristics and correlates of factors that contribute to poor long-term health outcomes.

### **Childhood Sexual Abuse and Telomere Length in Adulthood**

Results from the current study did not lend support to the first hypothesis that female twins exposed to CSA would have shorter mean TL compared to unexposed twins. Although this was not consistent with my hypothesis, these results are not uncommon within the recent literature (e.g., Glass et al., 2010; Keicolt-Glaser et al., 2011) and are consistent with the most recent report examining this association among a large sample of middle-aged females (Mason et al., 2015). The current study did have the advantage of a discordant MZ twin design, allowing me to control for genetic and shared environmental factors contributing to TL, but other unique environmental factors could have affected the relation between CSA and TL. For example, there are a plethora of other life experiences (e.g., exposure to abuse and/or violence after age 16, psychosocial stressors) that could have affected either twin, leading to similar mean TL within the current sample. Although we cannot control for all unique environmental factors that contribute to telomere attrition, future studies should bear in mind other stressors (both severe and common) that could impact an individual (e.g., abuse during adulthood).

I also examined differences in mean TL as a function of abuse severity (i.e., in some of the moderation models), but found no significant differences in mean TL across varying levels of severity. Only one study to date has examined the effects of CSA severity (operationalized as

sexual touching only versus physically and verbally abrasive abuse) on TL, finding that there was no effect of CSA – regardless of severity – on TL in a sample of middle-aged females (Mason et al., 2015). Given that this is the only study to do so, and that their measure of severity was different from the current study, it is difficult to know whether or not this null finding is to be expected. However, a recent study by Fergusson and colleagues (2013) followed a sample of individuals – exposed to CSA – over a 30-year period, finding that increases in CSA severity (via non-genital contact, genital contact, and intercourse) were significantly associated with an increased likelihood of individuals developing physical and mental health problems in adulthood. Since the effects of severity have an impact on physical health, it is possible that these effects extend to the molecular level, but there is currently no evidence to support this hypothesis. Nonetheless, the association between CSA severity and TL warrants further examination, particularly among more diverse, generalizable samples. Overall, in the context of previous findings, the present results suggest that any sexual abuse during childhood has an equivalent biological impact on the victim, as it pertains to middle-aged Caucasian females.

### **Social Support and Optimism as Moderators**

Results from linear mixed model analyses revealed several interesting findings. First, results from the model assessing only individuals exposed to CSA – and treating CSA as a continuous variable – revealed an interaction between CSA severity and social support. Specifically, as CSA severity increased, increases in social support were associated with decreases in mean TL. However, as CSA severity decreased, increases in social support were associated with increases in mean TL. This finding was contradictory to my original hypothesis. This finding could be due, in part, to low reliability for the social support scale ( $\alpha = .69$ ), which could lead to spurious results. Given the results from post-hoc analyses, it is possible that

combining the social support scales biased the findings, but it still does not fully explain why individuals exposed to more severe forms of CSA may be unaffected by social support.

One explanation for the unexpected interaction is that the type of support assessed with these few questions did not accurately capture the type of social support needed to serve as a protective factor at higher levels of CSA severity. The scale used in the current study was originally developed to assess multiple aspects of social support (i.e., instrumental and emotional); however, the scale is limited in its use of more broad questions regarding support (e.g., “express interest in how you are doing,” “make too many demands on you”). A study by Hyman and colleagues (2003) found that – for middle-aged females exposed to CSA – social support buffered the effects of CSA on psychological maladjustment; however, the strongest associations were found among self-esteem and appraisal support. Self-esteem support referred to support that made the individual feel as if they could positively compare themselves to others, while appraisal support referred to the individual being able to talk to others comfortably about their problems. Neither of these forms of support is accurately captured in the current study, and may explain why I did not find support for this particular hypothesis.

Another explanation for the current finding considers the impact of the most severe form of sexual abuse. Specifically, since intercourse is such a severe, invasive form of sexual abuse that the victim’s ability to engage in trusting social relationships may be severely inhibited and even detrimental, particularly when it originates from the family. For example, of the 24 individuals exposed to intercourse in the current sample, 11 identified the perpetrator as a relative and 12 identified the perpetrator as living in the same household during the time of abuse. It is possible that social support from relatives – even decades after the abuse has taken place – is counteractive to the victim, particularly if they were living with or nearby the victim

during childhood. In line with this hypothesis, it may be more appropriate to model social support as a mediator between CSA and TL. For example, a recent study by Herrenkohl and colleagues (2016) found no evidence for the moderating role of social support in the association between childhood abuse (including CSA) and adult physical and mental health outcomes. They did, however, find evidence for a mediating role of social support and suggest that childhood abuse severely inhibits the victim's ability to develop and maintain social relationships that are necessary for long-term physical and mental health. In sum, the present results suggest that social support may serve as a protective factor in the context of less severe forms of sexual abuse, but also can serve as a detrimental factor in the presence of more severe forms of abuse, perhaps if the perpetrator was someone who may be within the victim's support system.

The analyses examining optimism did not lend support for a moderating role of optimism in the association between CSA and mean TL. This was unexpected given the theoretical and empirical support for the association between optimism and TL (Schutte et al., 2016). However, a significant association between optimism and CSA was detected in preliminary analyses, such that individuals exposed to any CSA reported lower levels of optimism compared to individuals with no history of abuse. Thus, it is possible that the highest levels of optimism among exposed individuals in the current sample were not sufficient to serve a protective role in the association between CSA and TL. Another unexpected finding was that there was no association between social support and optimism within the current sample. As previously mentioned, the unreliable friends' social support items could have biased this correlation, but examination of bivariate correlations between optimism and social support assessing the two social support sub-scales separately revealed no significant associations.

Another explanation for our null findings regarding optimism is the time between abuse, assessment of optimism, and assessment of TL. Although the time between assessment of optimism and TL allows for the biological effects of optimism to become noticeable, it is possible that changes in optimism varied significantly between assessment time points. In addition, optimism could have been consistent across time points (i.e., between Time 1 and Time 5), but levels of optimism prior to Time 1 could have been much lower, and thus the biological effects of increased optimism over the course of the study would be negligible. This hypothesis, however, is difficult to test, as it would require repeated assessments over several decades. Nevertheless, this is the first study to examine the moderating role of optimism in the association between CSA and TL in adults and the present findings provide a glimpse into these associations.

### **Limitations**

Although the current study has several strengths (e.g., discordant MZ twin design) and adds valuable information to the existing literature examining the relation between CSA and TL in adults, there are several limitations to keep in mind when interpreting the findings. First, the uniqueness of the sample limits the generalizability of our findings. Twins are a unique sub-population that are not representative of the general population and may differ in their development from non-twin siblings. For example, it is possible that the unique closeness shared by twins may make them more likely to discuss certain experiences that in turn affect both twins. Another limitation of the current study is the small sample size. Although these smaller samples are not uncommon in the literature examining the association between CSA and TL (Oliveira et al., 2016), recent work with larger samples (e.g., Mason et al., 2015) has found no association

between CSA and TL, and has cited small samples as the reason for spurious findings within this literature.

The current study also was limited in its assessment of social support and optimism. As mentioned, post-hoc analyses revealed poor reliability for social support items from friends, which could have impacted the findings. In addition, the social support measure was an original measure and only consisted of five identical items assessing support from family and friends, respectively. It is possible that use of a more comprehensive, reliable measure would yield different findings. Further, although the scale assessing optimism was reliable and has previously demonstrated good reliability, it may be better to assess this construct (as well as social support) closer to the time of abuse. Although these constructs tend to be consistent throughout the lifespan, CSA is a traumatic event and many experiences have transpired since the assessment of these constructs, which could influence their effects on TL in the present study. Lastly, the retrospective nature of the study makes it difficult for us to account for unique environmental factors that each twin was exposed to throughout their lifespan. For example, the unexposed twin many have been exposed to other severe stressors leading to similar mean TL among twins, or exposed twins could have been exposed to other protective factors not assessed in the current study that could have contributed to similar mean TL among twins. Assessment of other life stressors would provide a more accurate depiction of the unique effects of CSA on TL in female adult twins.

## **Summary**

The present study adds to our understanding of the association between CSA and TL among middle-aged female twins, and addresses a gap in the literature by examining the role of protective factors in relation to TL. Although the current findings do not lend support for a direct



association between CSA and TL in female adult twins, social support does appear to play a moderating role in this association, albeit for women with the least severe type of CSA exposure. Given that this literature is still in its infancy, the examination of the role of protective factors in the association between CSA and TL among larger, representative samples will clarify the current findings. Nonetheless, the present findings advance our understanding of the biological effects of CSA and serve to inform prevention and intervention efforts for victims of sexual abuse.

## References

- Asok, A., Bernard, K., Roth, T. L., Rosen, J. B., & Dozier, M. (2013). Parental responsiveness moderates the association between early-life stress and reduced telomere length. *Development and Psychopathology*, 25, 577–585.
- Barger, S. D., & Cribbet, M. R. (2016). Social support sources matter: Increased cellular aging among adults with unsupportive spouses. *Biological Psychology*, 115, 43–49.
- Barker, D. J. P. (1990). The fetal and infant origins of adult disease. *British Medical Journal*, 301, 1111.
- Bates, D., Maechler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, 67, 1-48.
- Beach, S. R. H., Lei, K., Brody, G., Yu, T., & Philibert, R. (2014). Nonsupportive Parenting affects telomere length in young adulthood among african americans: Mediation through substance use. *Journal of Family Psychology*, 28, 967–972.
- Blackburn, E. H., Greider, C. W., & Szostak, J. W. (2006). Telomeres and telomerase: The path from maize, Tetrahymena and yeast to human cancer and aging. *Nature Medicine*, 12, 1133–1138.
- Blackburn, E. H. (2005). Telomeres and telomerase: Their mechanisms of action and the effects of altering their functions. *FEBS Letters*, 579, 859–862.
- Brody, G. H., Yu, T., Beach, S. R. H., & Philibert, R. R. A. (2015). Prevention effects ameliorate the prospective association between nonsupportive parenting and diminished telomere length. *Prevention Science*, 16, 171–180.
- Browne, A., Finkelhor, D., Baron, L., Briere, J., Ellison, J., Friedrich, W., & Ellen, M. (1986). Impact of child sexual abuse : A review of the research, 99, 66–77.

- Butchart, A., Phinney, H. A., Kahane, T., Mian, N., & Furness, T. (2006). Preventing child maltreatment: A guide to action and generating evidence. Geneva: World Health Organization and International Society for Prevention of Child Abuse and Neglect.
- Carroll, J. E., Roux, A. V. D., Fitzpatrick, A. L., & Seeman, T. (2013). Low social support is associated with shorter leukocyte telomere length in late life: Multi-ethnic study of atherosclerosis (MESA). *Psychosomatic Medicine*, 75, 1–15.
- Carver, C. S., & Scheier, M. F. (2014). Dispositional optimism. *Trends in Cognitive Sciences*, 18, 293–299.
- Cawthon, R. M. (2009). Telomere length measurement by a novel monochrome multiplex quantitative PCR method. *Nucleic Acids Research*, 37, 1-7.
- Child maltreatment*. (2016, September). Retrieved from <http://www.who.int/mediacentre/factsheets/fs150/en/>.
- Cohen, S., & Wills, T. A. (1985). Stress, social support , and the buffering hypothesis. *Psychological Bulletin*, 98, 310–357.
- Cook, R. D., & Weisberg, S. (1982). Residuals and influence in regression. New York, NY. Chapman and Hall.
- Diaz, V. A., Mainous, A. G., Everett, C. J., Schoepf, U. J., Codd, V., & Samanii, N. J. (2010). Effect of healthy lifestyle behaviors on the association between leukocyte telomere length and coronary artery calcium. *American Journal of Cardiology*, 106, 659–663.
- Eisenberger, N. I., Taylor, S. E., Gable, S. L., Hilmert, C. J., & Lieberman, M. D. (2007). Neural pathways link social support to attenuated neuroendocrine stress responses, 35, 1601–1612.
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of

the leading causes of death in adults: The adverse childhood experiences (ACE) study.

*American Journal of Preventive Medicine*, 14, 245–258.

Fergusson, D. M., McLeod, G. F. H., & Horwood, L. J. (2013). Childhood sexual abuse and adult developmental outcomes: Findings from a 30-year longitudinal study in New Zealand. *Child Abuse and Neglect*, 37, 664-674.

Finkelhor, D., Shattuck, A., Turner, H. A., & Hamby, S. L. (2014). The lifetime prevalence of child sexual abuse and sexual assault assessed in late adolescence. *Journal of Adolescent Health*, 55, 329–333.

Glass, D., Parts, L., Knowles, D., Aviv, A., & Spector, T. D. (2010). No correlation between childhood maltreatment and telomere length. *Biological Psychiatry*, 68, e21–e22.

Graignic-Philippe, R., Dayan, J., Chokron, S., Jacquet, A. Y., & Tordjman, S. (2014). Effects of prenatal stress on fetal and child development: A critical literature review. *Neuroscience and Biobehavioral Reviews*, 43, 137–162.

Haycock, P. C., Heydon, E. E., Kaptoge, S., Butterworth, A. S., Thompson, A., & Willeit, P. (2014). Leucocyte telomere length and risk of cardiovascular disease: Systematic review and meta-analysis. *British Medical Journal*, 349, 1-11.

Herrenkohl, T. I., Jung, H., Klika, J. B., Mason, A. W., Brown, E. B., Leeb, R. T., & Herrenkohl, R. C. (2016). Mediating and moderating effects of social support in the study of child abuse and adult physical and mental health. *American Journal of Orthopsychiatry*, 86, 573-583.

Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7, 1-20.

Hulme, P. A. (2004). Theoretical perspectives on the health problems of adults who experienced childhood sexual abuse. *Issues in Mental Health Nursing*, 25, 339–361.

- Hyman, S. M., Gold, S. N., & Cott, M. A. (2003). Forms of social support that moderate PTSD in childhood sexual abuse survivors. *Journal of Family Violence, 18*, 295-300.
- Ikeda, A., Schwartz, J., Peters, J. L., Baccarelli, A. A., Hoxha, M., Dioni, L., ... & Kubzansky, L. D. (2014). Pessimistic orientation in relation to telomere length in older men: The VA normative aging study. *Psychoneuroendocrinology, 42*, 68-76.
- Irish, L., Kobayashi, I., & Delahanty, D. L. (2010). Long-term physical health consequences of childhood sexual abuse: A meta-analytic review. *Journal of Pediatric Psychology, 35*, 450-461.
- Jobin, J., Wrosch, C., & Scheier, M. F. (2014). Associations between dispositional optimism and diurnal cortisol in a community sample: When stress is perceived as higher than normal. *Health Psychology, 33*, 382-391.
- Jones, T. M., Hill, K. G., Epstein, M., Lee, J. O., Hawkins, J. D., & Catalano, R. F. (2016). Understanding the interplay of individual and social-developmental factors in the progression of substance use and mental health from childhood to adulthood. *Development and Psychopathology, 28*, 721-741.
- Kendler, K. S., & Prescott, C. A. (2006). *Genes, environment, and psychopathology: Understanding the causes of psychiatric and substance use disorders*. New York, NY: Guilford Press.
- Kiecolt-Glaser, J. K., Gouin, J., Weng, N., Malarkey, W. B., Beversdorf, D. Q., & Glaser, R. (2011). Childhood adversity heightens the impact of later-life caregiving stress on telomere length and inflammation. *Psychosomatic Medicine, 73*, 16-22.
- Kubzansky, L. D., Sparrow, D., Vokonas, P., & Kawachi, I. (2001). Is the glass half empty or half full? A prospective study of optimism and coronary heart disease in the normative aging study. *Psychosomatic Medicine, 63*, 910-916.

- Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2016). lmerTest: Tests in linear mixed effects models. R package version 2.0-33. <https://CRAN.R-project.org/package=lmerTest>.
- Maniglio, R. (2009). The impact of child sexual abuse on health: A systematic review of reviews. *Clinical Psychology Review, 29*, 647–657.
- Mason, S. M., Prescott, J., Tworoger, S. S., DeVivo, I., & Rich-Edwards, J. W. (2015). Childhood physical and sexual abuse history and leukocyte telomere length among women in middle adulthood. *PloS One, 10*, 1-12.
- Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology, 22*, 491–495.
- Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137*, 959–997.
- Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–524.
- Mullen, P. E., Martin, J. L., Anderson, J. E., Romans, S. E., & Herbison, G. P. (1993). Childhood sexual abuse and mental health in adult life. *British Journal of Psychiatry, 163*, 721-732.
- O'Donovan, A., Lin, J., Dhabhar, F. S., Wolkowitz, O., Tillie, J. M., Blackburn, E., & Epel, E. (2009). Pessimism correlates with leukocyte telomere shortness and elevated interleukin-6 in post-menopausal women. *Brain, Behavior, and Immunity, 23*, 446–449.
- O'Donovan, A., Epel, E., Lin, J., Wolkowitz, O., Cohen, B., Metzler, T., ... & Thomas, C. (2011). Childhood trauma associated with short leukocyte telomere length in post-traumatic stress disorder. *70*, 465–471.

- Oberlander, T. F., Weinberg, J., Papsdorf, M., Grunau, R., Misri, S., & Devlin, A. M. (2008). Prenatal exposure to maternal depression, neonatal methylation of human glucocorticoid receptor gene (NR3C1) and infant cortisol stress responses. *Epigenetics*, 3, 97–106.
- Oliveira, B., Zunzunegui, M. V., Quinlan, J., Fahmi, H., Tu, M. T., & Guerra, R. (2015). Systematic review of the association between chronic social stress and telomere length: A life course perspective. *Ageing Research Reviews*, 26, 37–52.
- Price, L. H., Kao, H. T., Burgers, D. E., Carpenter, L. L., & Tyrka, A. R. (2013). Telomeres and early-life stress: An overview. *Biological Psychiatry*, 73, 15–23.
- Rasmussen, H. N., Scheier, M. F., & Greenhouse, J. B. (2010). Optimism and physical health: A meta-analytic review. *Annals of Behavioral Medicine*, 37, 239–256.
- Rius-Ottenheim, N., Houben, J. M. J., Kromhout, D., Kafatos, A., Van Der Mast, R. C., Zitman, F. G., ... & Giltay, E. J. (2012). Telomere length and mental well-being in elderly men from the Netherlands and Greece. *Behavior Genetics*, 42, 278–286.
- Rode, L., Nordestgaard, B. G., & Bojesen, S. E. (2015). Peripheral blood leukocyte telomere length and mortality among 64,637 individuals from the general population. *Journal of the National Cancer Institute*, 107, 1-8.
- RStudio Team (2016). RStudio: Integrated Development for R. RStudio, Inc., Boston, MA.  
<http://www.rstudio.com/>.
- Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: assessment and implications of generalized outcome expectancies. *Health psychology*, 4, 219.
- Schutte, N. S., Palanisamy, S. K. A., & McFarlane, J. R. (2016). The relationship between positive psychological characteristics and longer telomeres. *Psychology & Health*, 446, 1–15.

- Shalev, I., Moffitt, T. E., Sugden, K., Williams, B., Houts, R. M., Danese, A., ... & Caspi, A. (2013). Exposure to violence during childhood is associated with telomere erosion from 5 to 10 years of age: A longitudinal study, *18*, 576–581.
- Shumaker, S. A., & Czajkowski, S. M. (1994). *Social Support and Cardiovascular Disease*. Shumaker, S. A., & Czajkowski, S. M. (Eds.). New York, NY: Plenum.
- Smith, T. W., & MacKenzie, J. (2006). Personality and risk of physical illness. *Annual Review of Clinical Psychology*, *2*, 435–467.
- Smith, T. W. (2006). Personality as risk and resilience in physical health. *Current Directions in Psychological Science*, *15*, 227–231.
- Spitzer, R. L., Williams, J. B., & Gibbon, M. (1987). *Structured Clinical Interview for DSM-III-R*. New York: New York State Psychiatric Institute Biometrics Research Department.
- Starkweather, A. R., Alhaeeri, A. A., Montpetit, A., Brumelle, J., Filler, K., Montpetit, M., ... & Jackson-Cook, C. K. (2014). An integrative review of factors associated with telomere length and implications for biobehavioral research. *Nursing Research*, *63*, 36-50.
- Surtees, P. G., Wainwright, N. W. J., Pooley, K. A., Luben, R. N., Khaw, K. T., Easton, D. F., & Dunning, A. M. (2011). Life stress, emotional health, and mean telomere length in the European prospective investigation into cancer (EPIC)-Norfolk population study. *Journals of Gerontology*, *66*, 1152–1162.
- Talge, N. M., Neal, C., & Glover, V. (2007). Antenatal maternal stress and long-term effects on child neurodevelopment: How and why? *Journal of Child Psychology and Psychiatry*, *48*, 245–261.
- Taylor, S. E. (2010). Mechanisms linking early life stress to adult health outcomes. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 8507–8512.

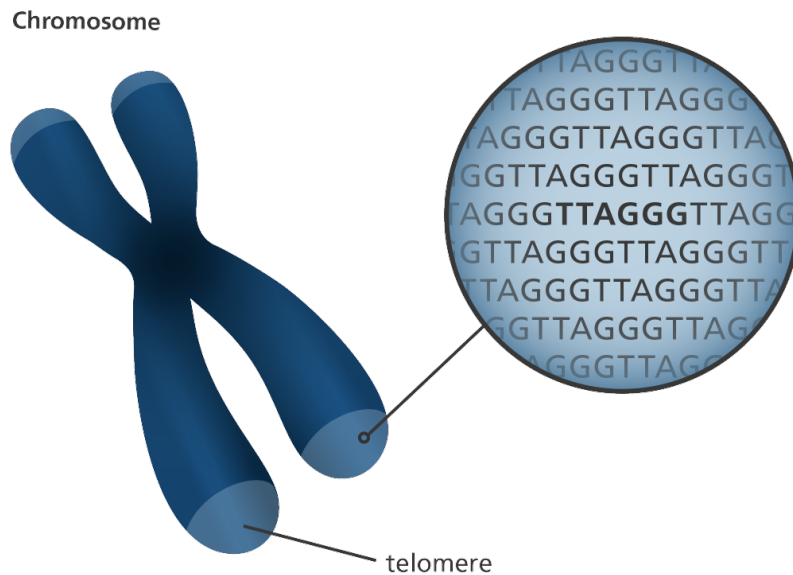


- Tindle, H. A., Chang, Y. F., Kuller, L. H., Manson, J. E., Robinson, J. G., Rosal, M. C., ... & Matthews, K. A. (2009). Optimism, cynical hostility, and incident coronary heart disease and mortality in the women's health initiative. *Circulation*, *120*, 656–662.
- Tyrka, A. R., Price, L. H., Kao, H., Marsella, S. A., & Carpenter, L. L. (2011). Childhood maltreatment and telomere shortening: Preliminary support for an effect of early stress on cellular aging, *67*, 531–534.
- Uchino, B. N., Cawthon, R. M., Smith, T. W., Light, K. C., McKenzie, J., Carlisle, M., ... & Bowen, K. (2012). Social relationships and health: Is feeling positive, negative, or both (ambivalent) about your social ties related to telomeres?, *31*, 789–796.
- Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine*, *29*, 377–387.
- Wegman, H. L., & Stetler, C. (2009). A meta-analytic review of the effects of childhood abuse on medical outcomes in adulthood. *Psychosomatic Medicine*, *71*, 805–812.
- Wright, M. O. D., Masten, A. S., & Narayan, A. J. (2013). Resilience processes in development: Four waves of research on positive adaptation in the context of adversity. In S. Goldstein & R. B. Brooks (Eds.), *Handbook of Resilience of Children* (2nd ed., pp. 15–37). New York: Springer.
- Zalli, A., Carvalho, L. A., Lin, J., Hamer, M., Erusalimsky, J. D., Blackburn, E. H., & Steptoe, A. (2014). Shorter telomeres with high telomerase activity are associated with raised allostatic load and impoverished psychosocial resources. *Proceedings of the National Academy of Sciences of the United States of America*, *111*, 4519–4524.

### Vita

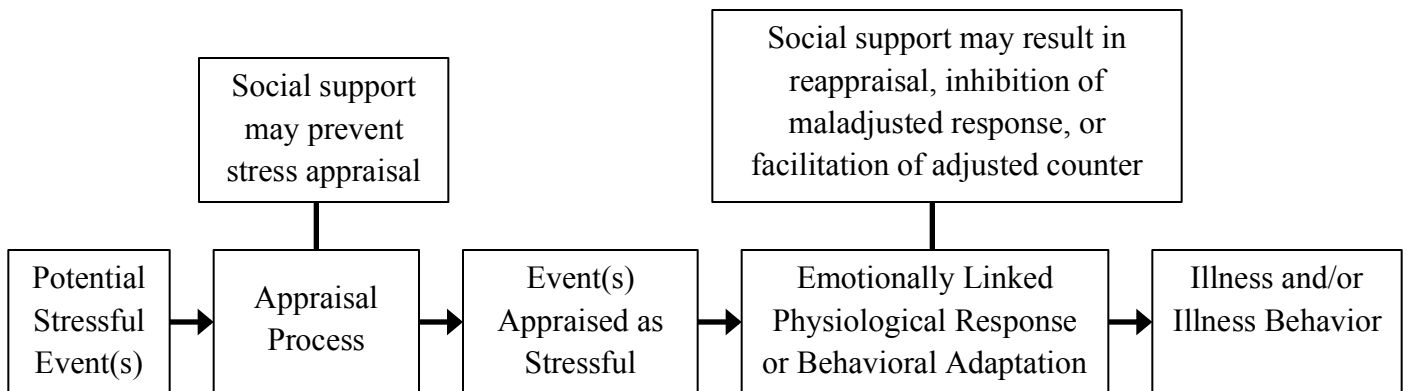
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**Figure 1.** Location of Telomeres on a Human Chromosome



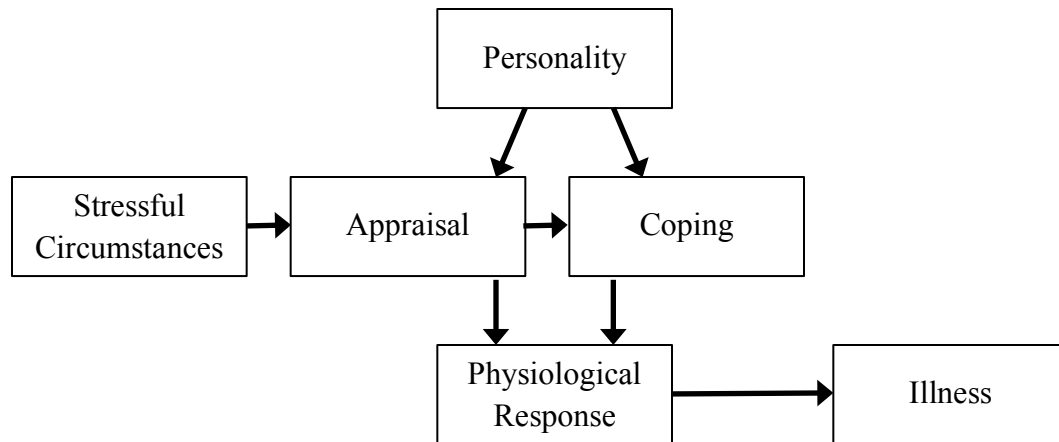
*Figure 1.* Telomeres are repeat strands of DNA (in humans, TTAGGG) located at the end of chromosomes that serve to protect genetic data during DNA replication. Because of the “end replication problem” telomeres degrade over time until they reach a critically short length, which signals cellular senescence. Thus, telomeres prevent DNA damage and serve as a biological marker of cellular aging. Image credit: Genome Research Limited.

**Figure 2.** Social Support and the Buffering Hypothesis Model



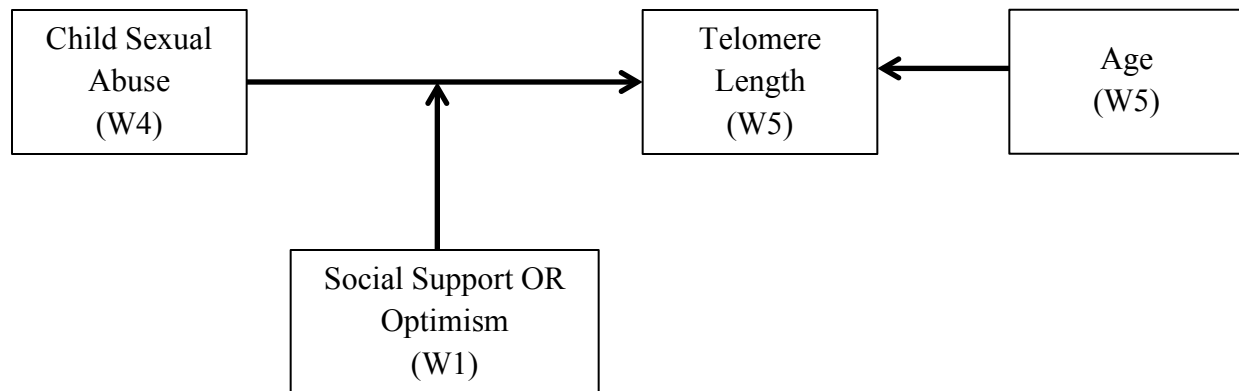
*Figure 2.* Cohen and Wills' (1985) model depicting two points at which social support may influence the link between stress and later illness. Copyright 1985 by the American Psychological Association.

**Figure 3.** Interactional Stress Moderation Model



*Figure 3.* Smith's (2006) model depicting how personality may affect the coping and/or appraisal process following a stressful event, which affects the physiological response that ultimately contributes to illness. Copyright 2006 by the Association for Psychological Science.

**Figure 4.** Hypothesized Model for Hypotheses 2 and 3.



*Note.* Child sexual abuse was assessed retrospectively at Wave 4. Social support and optimism were assessed in separate models.